THE VALUE OF EYE SYMPTOMS IN THE DIAGNOSIS OF GENERAL DISEASES*

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It is often said that the ophthalmologist is merely a specialist. This, however, I hope to be able to contradict. He is and must be—if he really wishes to fill his place—a general physician just as much as the general practitioner. Sir Archibald Garrod impressed this upon us in his speech at Oxford in 1927. His subject was the connexion between internal medicine and ophthalmology.

"What is the difference between a specialist and a quack? Just one little thing. The real specialist thinks always in a general way. He therefore is able to understand the medical process in question. The quack knows merely the symptoms, and is therefore in danger of becoming a symptomatician."

Even if the eye is the most differentiated organ of the body, it is nevertheless merely a single, although a very important, part of the united organs constituting the animal body. In my lecture I hope to prove that a series of symptoms in the eye are due to diseases somewhere else in the body. Thinking of the histology of the eye, it must be evident that the pathology of the central nervous system is rich in eye symptoms. The visual fibre system forms a continuity of axons from the ganglion cells of the retina to its ending in the pole of the occipital lobe. On its way it is in touch with a lot of very important nerve nuclei. Because of this any pathological process here is sure to show itself in the visual organ. In passing, I must call attention to the transient pareses of the eye muscles—especially of the abducens—in tabes, lues cerebri, encephalitis, and disseminated sclerosis. Still more important as a very early symptom in late syphilitic disease is the Argyll Robertson pupil reaction. I think this is the first ocular eye symptom the young medical student is taught when entering a general medical ward. It is superfluous to mention that an exact examination of the pupil reaction may often prevent the overlooking of tabes in recurrent attacks of colic and vomiting.

DISSEMINATED SCLEROSIS

Charcot taught that the cardinal symptoms of disseminated sclerosis were scanning speech, intention tremor, and nystagmus. No greater obstacle to the early diagnosis of this common disease could be met with than a reliance on this statement. This "classical" triad is found complete, according to Marburg, in only about 15 per cent. of all cases. Most of them are immature, "*formes frustes*." In the *Nordisk Medicinsk Tidsskrift* (1929) I published a paper about the eye symptoms of this affection. I emphasized therein that if we still were to accept a diagnostic triad this ought to be: (1) pronounced fatigue following insignificant body exercises and at the same time accentuation of symptoms already present (I hold that the symptom of Uhthoff—passing reduction of vision after a short but quick walk—must come in this class); (2) increase of the tendon reflexes; and (3) passing eye symptoms, frequently with a recurring tendency. W. A. Adie reaches the same conclusion in his excellent work in the *British Medical Journal* (1932). Acute scotomatous retrobulbar neuritis is the most important eye symptom in this disease. Traquair characterizes this neuritis, in his noteworthy Doyne Memorial Lecture in 1923, in the following way. It commences quickly, and is of a changing tendency during its run, ending almost with complete cure of the initial attack. However, it is apt to recur. As a rule it is one-sided, although in my experience I have fre-

quently found it to be bilateral. Not uncommonly this neuritis appears years before the other symptoms of this insidious disease: in the literature there are cases up to eighteen years. The scotoma in this neuritis is always central. In this it differs from the intoxication amblyopias, where the scotomas always commence paracentrally. It may arise not only in an acute but also in a subacute and insidious chronic form. Authors differ widely as regards its percentage of incidence. The lowest figure I have found is 13 per cent., the highest 70 per cent. Personally, I believe 50 per cent. to be the right figure.

Passing palsies of the eye muscles not uncommonly appear, either isolated or together with other symptoms, in multiple sclerosis. They are stated to occur in 25 to 45 per cent. of cases. They may be one-sided or bilateral. Most frequently we find paralysis of the abducens, not uncommonly coupled with paresis of the facial nerve. The nuclei of these nerves are located, as you remember, below the floor of the fourth ventricle. Their root fibres run together with the pyramidal fibres through the pons, which is the common site of selection of the sclerotic plaques. Therefore we frequently notice phenomena from these. Any facial palsy, where all the chief branches are engaged, should always make the physician think of a possible disseminated sclerosis as the underlying cause. Without doubt, a lot of the so-called "rheumatic" facial and abducens palsies are due to this disease in its earliest stages. At least such an origin ought to be kept in mind in all cases of passing eye-muscle paralyses, especially when they are coupled with "rheumatism" somewhere else in the body. Again, we must never forget tabes as a cause. The oculomotor and trochlear nerves are less often attacked by the multiple sclerosis. Their nuclei are situated below the bottom of the aqueductus Sylvii, and thus are above the pons. Recurrent ptosis, however, is often observed together with paresis of the internal rectus muscles. In tabes an isolated paresis of the fourth nerve is not uncommon as a very early symptom.

EYE SIGNS IN SYPHILIS

Argyll Robertson pupils were previously considered as a sure indication of an earlier metaluetic infection. At the present time we know that this phenomenon may arise in the different varieties of encephalitis. This disease is itself characterized by a number of eye symptoms: ptosis, nystagmus, and paralysis of the accommodation are particularly common, and these eye symptoms may make the diagnosis infinitely easier. Time does not allow me to go more fully into details of the pupillary symptoms in general diseases. I merely note that, according to Uhthoff, in more than 80 per cent. of tabetic cases and in general paralysis in about 70 per cent. there is the characteristic reaction of Argyll Robertson. With lues cerebro-spinalis, however, it is comparatively seldom noticed. In this disease an internal ophthalmoplegia, combined with palsies of other cerebral nerves, is more often found. Defects of the field of vision—mostly hemianopic—are also commonly seen in cerebral syphilis. A distinct anisocoria is always bound to attract the attention of the physician to a previous infection with the *Spirochaeta pallida*, or—as Sir William Macewen showed so early as 1884—if this is excluded, to an increasing brain pressure, especially a growth in the temporal lobe. On the contrary, an insignificant difference of the pupils with normal reaction is not rare. Probably this is brought about by traumata

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during birth. Irregularities of the pupillary margin—without synechiae—have in earlier days been considered important for the diagnosis of cerebral lues. The importance of this has been overrated, except when it is coupled with disturbances of the pupillary reaction.

Spinal miosis in tabes is frequently noticed owing to affection of the sympathetic in the spinal medulla. It is caused by secondary contraction of the sphincter pupillae, and may be very pronounced. As a rule it is one-sided. In this way it differs from the contraction observed in certain chronic cases of poisoning—especially morphinism. In this latter, however, there is always a normal pupil reaction. In cases of one-sided miosis Horner's syndrome must always be remembered. As you know, this is associated with tumours in the cervical region or even lower—for instance, aneurysm of the aorta—causing paralysis of the sympathetic.

EYE SIGNS IN INFECTIONS

Not only in lues cerebri and metaluetic diseases is the pupillary reaction of great diagnostic importance, but also in a number of other brain diseases. The same is the case with an ophthalmoplegia interna. With a feverish patient a paralysis of the pupil reaction as well as the accommodation may be the single sign of a commencing meningitis. In most of such cases, however, other cerebral nerves will also be affected. These pareses we find most often in luetic and tuberculous inflammation, and more seldom in otogenic meningitis.

I have already mentioned that in certain chronic cases of poisoning—such as morphinism—the size of the pupils may be altered, while their reaction remains normal. In a number of acute toxæmias in febrile diseases there may be paralysis of the pupil. Especially well known is the post-diphtheritic paralysis of accommodation. After an influenza the same occurs, but not so commonly. Both conditions give rise to a bilateral palsy. Cyclitis is not infrequently observed after influenza. Here I may mention that a number of acute infectious diseases, during their febrile stage, are accompanied by a more or less pronounced conjunctivitis. During a measles epidemic, conjunctivitis is a most important diagnostic feature. It may be the first prodromal symptom. Botulism, and meat and fish poisoning, may all be followed by paralysis of the pupil and of other eye muscles. This is also the case with grave gastro-enteritis and paratyphoid fever. The picture is similar to the one seen in acute general atropine poisoning.

METABOLIC DISTURBANCES

It is well known that diabetes mellitus during its progress may be accompanied by passing myopia as well as by transient hypermetropia. Every sixth diabetic has, as a rule, eye complications. Iritis with hypopyon occurs seldom. It is, as regards the general condition, a very severe symptom. All cases of iritis ought, therefore, to be examined for sugar. Sigurd Hagen is probably the first to have emphasized strongly that a transitory hypermetropia never arises in the case of an untreated diabetic. On the other hand, a transitory near-sightedness is not infrequently the very symptom causing the detection of a previously overlooked diabetes. Of special interest is the following case. The myopia reached here the highest figure I have noticed, afterwards completely passing away:

On June 19th, 1920, a gentleman, 67 years of age, called on me to get spectacles. With + 3.5 the vision was 6/4 both eyes. On October 12th, 1921, he returned again, because for the last eight days the vision had faded for distance. With his spectacles for near vision he was just able to read. Used + 6.0. Upon examination normal externa and interna were found. The left eye showed emmetropia, the right - 1.5 myopia. A pronounced positive sugar reaction was found in the urine—also traces of albumin. He was advised to see a diabetes

specialist. During a short space of time a psychosis developed with sensations of inferiority. He died two years afterwards from apoplexy. In the meantime, the myopia had entirely disappeared, and he grew hypermetropic again.

Time does not allow me to enter into the different theories about these passing anomalies. With reference to the myopia I refer to the work of Ask, published in the *Biochemische Zeitschrift* (1913). As to the transitory hypermetropia, I draw attention to Granström's great work in the *Acta Ophthalmologica* (1933). He seeks to prove that a sodium chloride retention is to be found in the lens in diabetics, causing a retention of fluid and alteration of the index. Generally speaking, chemical alterations of the blood—speaking broadly—may be followed by eye symptoms. Of the greatest importance to the general practitioner is, I think, chronic conjunctivitis. Without using tonics we often are not able to cure this type of eye trouble, and there is no doubt that many an eye doctor would have better results in his treatment of conjunctivitis if he were to investigate the colour index of the blood. A remarkable symptom caused by anaemia in chronic conjunctivitis, as well as the fairly common asthenopia, is a slight oedema of the lids. This is mostly pronounced in the morning, disappearing later in the day. This peculiarity is probably caused by the massage effect of repeated blinking.

During the last decade a great many works have been published on the subject of alteration in the blood as an underlying cause of eye diseases. By using the Reid Hunt acetonitril reaction it has been held that certain eye troubles (for example, glaucoma) are due to a hyperiodaemia. This might be of great practical consequence. We should in such cases be most careful in the use of iodides and thyroid extract or other hormone preparations in our treatment of hypertonia and arteriosclerosis. Lagrange is the first to have declared that glaucoma is no local disease, but merely an evidence of a constitutional state. Magitot and Angelucci have formed the opinion that all glaucoma patients are people with diseased vessels. They term such a person "*un vasculaire*," an individual with a disturbed capillary circuit. Others, such as Eppinger, Leube, von Hesse, and Meller, have sought the cause in a disturbance of the equilibrium between the sympathetic and the parasympathetic. They support the opinion by the fact that psychic disturbances aggravate an existing glaucoma, also that glaucoma sometimes, like ophthalmic migraine, improves considerably when the patient is put on a salt-poor diet and gets sufficient exercise to develop free perspiration.

CHANGES IN THE RETINA

Taking the diseases of the retina, we find that in no small percentage they are due to general disease, not uncommonly a disease of the blood. An orbital haemorrhage in infants and with black-red discoloration of conjunctiva and exophthalmos is not infrequently the first sign of a Möller-Barlow's disease, scurvy, or haemophilia. An ophthalmoscopic examination will very often disclose a retinal haemorrhage. All things considered, every haemorrhagic diathesis will tend towards bleeding in the external as well as the internal parts of the eye. Haemorrhages of the retina often appear with purpuric diseases, generally taken. From daily life we know that persons exist who are liable to haemorrhages into the skin after small traumatism. For this reason many a young girl at a dance resents what she considers too brutal a grip from her partner! However, if such a tendency is present in the retinal vessels the results are likely to be much more serious.

Pernicious anaemia is the typical blood disease generally associated with retinal alterations, in contrast to the secondary anaemias. These almost never lead to such. Here

I except the very rare amauroses and amblyopias following in the wake of heavy losses of blood. They are generally seen after abortions and haematemeses—much more infrequently after a normal birth. Their clinical picture is the same as that of embolism of the central artery of the retina. At the peak of pernicious anaemia retinal haemorrhages are very rarely lacking. White degenerative spots are also commonly noted in the oedematous retina. The finding of these alterations is not only of diagnostic but also of prognostic importance. They may disappear without trace when the patient recovers by the use of the liver diet of Minot. In leukaemias—both the myelogenous and the lymphatic—there may be characteristic alterations of the eye. In the lymphatic type as well as in pseudo-leukaemia we may have orbital tumours of lymphatic tissue. For this reason all orbital tumours demand an exact examination of the blood. As you know, the retina often reveals in leukaemia a characteristic light colour, thus contrasting with polycythaemia and congenital heart failure. Any fever of doubtful origin calls for ophthalmoscopy. I just mention the diagnosis of septicaemia, which is so very often difficult. Septic retinitis will, in these cases, make the diagnosis absolutely conclusive. According to the literature it should be observed in about 35 per cent. of all cases. More than one case of malignant endocarditis has been diagnosed by aid of the ophthalmoscope.

DIABETES AND NEPHRITIS

Two very common diseases in which the finding of retinal haemorrhages and exudative spots is of the greatest importance—not only in the diagnosis but also as regards prognosis—are nephritis and diabetes. The double-sidedness of the affection is characteristic of the retinal picture. As a rule it is more developed in one eye than in the other. The papilla shares in the affection to a greater or less extent. In the retina are to be seen oedema, scattered haemorrhages, and spots of white exudates. These last are often arranged like a star in the macular region. The retinitis in nephritis and diabetes is often without any symptom in its earliest stages. In this regard it is different from the usual optic neuritis, where the vision fails very rapidly on account of a central scotoma. Therefore ophthalmoscopy is of such great importance in any case of nephritis, and especially in the chronic cases. In particular, the retinitis in pregnancy may for a long time lack subjective symptoms. Upon examining 8,400 pregnant women Ingolf Schiötz found forty cases of retinitis gravidarum. Of these twenty-seven had chronic nephritis. Several of these had no subjective symptoms. Their vision was 6/6 or about 6/6. The prognosis in retinitis gravidarum, he found, is good, or at least fairly good. The alterations of the retina generally disappear entirely after birth or when an evacuation of the uterus has taken place. On the contrary, a usual retinitis albuminurica is a very bad prognostic sign. Elaborate statistics from large clinics have shown that only 10 per cent. of the affected patients are still alive more than two years after the appearance of the retinitis. And the prognosis is even worse the younger the individual is. According to Ascher the same may be said about the retinitis in diabetics. This, like the iritis, appears only in grave cases.

I must point out that even if the diagnosis of such a neuro-retinitis is easy in some cases, it may in some present very great difficulties. The papillitis may be so developed that the displacement of the papilla may be equal to the choked disk in a cerebral tumour. As these cases not uncommonly may show a star figure at the macula, even the most expert ophthalmologist may go wrong. As in both cases albuminuria may be present, and this periodically even may be absent in granulated kidney, the diagnosis may prove more difficult. Regard-

ing the differential diagnosis, a very exact functional examination is of vital importance. In a real choked disk the vision is, to begin with, less reduced than would be expected in view of the very pronounced ophthalmoscopic picture. With inflammation of the optic nerve the vision is quickly reduced on account of a central scotoma.

The pseudo-neuritis of hypermetropia must always be kept in mind. There is no reduction of vision and no central scotoma. Bleedings in the actual pupillary tissue and the surrounding retina speak for choked disk and against pseudo-neuritis.

I cannot here enter further into a description of embolism and thrombosis of the central retinal vessels; I just want to point out that in more than one case the disease apparently starts with these affections. The ophthalmologist often chances on this condition in an elderly patient calling to obtain reading glasses. An exact examination of his urine, heart, and blood pressure is required in such a case. It cannot be emphasized too strongly that an optic neuritis must never be taken for a *morbus sui generis*. This also applies to choked disk, in which latter condition we must remember that more than 80 per cent. are due to an intracranial growth.

HEMIANOPIA

Although hemianopia has been known for a period of about 150 years, its true significance has only been realized since 1855. Barthelomeo Panizza of Padua then proved clinically, pathologically-anatomically, and experimentally that the location of the vision centre is the cortex of the occipital lobe. I cannot here enter into the importance of the interpretation of the different types of hemianopias with reference to a focal diagnosis; I am merely going to mention that the bitemporal hemianopic scotomata are a very early sign of tumours of the hypophysis. I take the occasion to call attention to the excellent work of Traquair, regarding the interpretation of such scotomata. I must, however, be permitted to mention, very shortly, two cases from my own practice, where an exact scotoma investigation decided an otherwise difficult diagnosis.

A very hypochondriacal business man in his sixties had for a number of years been a worry to his doctor. One day he came to me telling me that he—*mirabile dictu*—was unable to read types of ordinary size, while, on the contrary, he could read the smallest print just as well as before. He dated his troubles from a passing blindness lasting for about one minute three weeks before. Normal external and internal conditions were found. Vision 6/4 O.U. The peripheral field completely normal. Examined with the scotometer of Haitz a right-sided wedge-shaped hemianopic scotoma was found. It was about 5 degrees in length, and lay with its edge only one degree from the fixation point. It was absolute for colours and relative for white. Upon letting the intelligent man read from right to left, he did so to his own great astonishment.

Here we have a case of the macular hemianopic reading disturbance of Wildbrand. Larger retinal pictures—in this case printed matter—are covered by the scotoma. Small retinal pictures are recognized between the fixation point and the edge of the scotoma facing them. The case shows how careful one must be with the diagnosis of neurasthenia and hypochondria. But for the field examination—with small objects—the fresh complaints of the patient might easily have been indexed among his usual "nervous" ones. In this case, I think, a punctate bleeding was really present in the left cuneus. A few months later he suffered from a new passing amaurosis, showing considerably greater hemianopia.

In the next case I am going to mention mental disease might easily have been diagnosed but for the examination of the ophthalmologist.

A painter, 54 years of age, was sent to me by a county colleague in March, 1920. It was stated that he had had hallucinations for a period of fourteen days after a short

unconsciousness. He imagined he saw the left part of the room filled with a number of people, whom he knew well and could name. He himself had, however, an idea that the whole thing was a "delusion"! He admitted abuse of spirits but denied lues. Upon investigation the eyes were normal, externally and internally. Vision with +1.5, 6/6 O.U. Tension 5/5.5 O.U. By perimetry a typical homonymous hemianopia was found. The entire left half of the fields had disappeared, with only a large rest for the macula left.

The cause of the hemianopia, as well as of his well-developed hallucinations, is one and the same: a focus of some kind in the right temporal lobe. This brain lesion must be subcortically located. It must be so placed that it produces a state of irritation both of his optical centre of memory as well as of his association province. It is further necessary that the cortex cerebri must be undamaged, as it is able to transfer the hallucinations to the consciousness of the patient. Before passing from the subject of the diagnostic value of the hemianopias I must merely mention that quite a few authors (for example, Ingolf Schiötz and Bauer) have demonstrated that passing homonymous hemianopias may arise in a number of auto-intoxications, especially the nephritis in pregnancy.

NYSTAGMUS

In the beginning of my paper I mentioned nystagmus as a link of Charcot's triad in the diagnosis of disseminated sclerosis. Marburg holds that real nystagmus, however, is only seen in at most 12 per cent. of all cases. Nystagmoidal tremblings are, according to Bárány, seen in 60 per cent. of normal people when looking extremely sideways. But even if this symptom is of no especial importance in the diagnosis of the above-mentioned disease, it is, however, as expressed by the Belgian Coppez, "*Une importance considérable en otologie, en neurologie et en hygiène générale.*" When he does not add "*en ophtalmologie*," although it is an eye symptom, it is in my opinion to emphasize that the specialist must never be one-sided! He must never lose sight of the organism in favour of the organ. The otologist Adrian de Kleijn of Utrecht is probably the man who has done the most valuable work on nystagmus next to Robert Bárány and Mario Camis. He meant to have a good reason for sacrificing two years in the study of ophthalmology before becoming purely an otologist.

This is not the place to enter upon the large subject of general medicine. I merely mention that in this domain the British surgeons have produced works of the highest lasting value. Thus the English doctor Gillot, in Sheffield in the year 1858, described "miner's nystagmus"—that is, three years ahead of the Belgian ophthalmologist Deconde. As we in Norway have no coal mines I have not personally seen this disease. Later on, very important studies on this subject were published in different countries. From the German side I only mention Johannes Ohm; from the British, Maitland Ramsay, Priestley Smith, sen., Elsworth, Stanley Percival, and Maurice Bartholomew. Every one of them has held that it is seen particularly with bad mine hygiene. In this connexion Percival's statement regarding blue-blindness preceding the nystagmus is of the utmost hygienic importance. That shock releases a latent nystagmus was demonstrated during the world war by Elliot and Stirling. Priestley Smith has shown that certain poisons (for example, iodoform and morphine) may play a part. This also indicated that the cerebrum must have some nystagmogenous influence. The fact that it is also seen in febrile diseases points in the same direction. Before leaving nystagmus I cannot but mention the great credit due to Bárány by the finding of caloric nystagmus in the diagnosis of a number of brain diseases, especially acoustic tumours. I also call your attention to the fact that Igersheimer and Heine have found that in not a few cases of congenital nystagmus the Wassermann reaction is positive. They

hold that vigorous antisyphilitic treatment has influenced this nystagmus in a favourable direction.

EYE SYMPTOMS IN POISONING

Time prevents me from entering into the eye symptoms which follow a number of poisonings. I merely mention a few points about santonin poisoning. Here the ophthalmologist may happen to fill the important role as legal-physician. In the case of such poisoning—as also with misuse of digitalis—xanthopsia not infrequently appears. Less well known to most of you may possibly be that Bruneche has described hallucinations of red vision, rhodopsia, with this poisoning: "Everything is floating in blood"! At the same time the poisoned person describes terrible murder scenes!

Acute poisoning from methyl alcohol is well known in the so-called "dry" countries. Acute gastro-enteritis supervenes—often some little time after the drinking. In addition to this a rapidly developing amaurosis, resulting from an acute degeneration of the ganglion cells of the retina, and a secondary atrophy of the optic nerve, is often observed. In the most pronounced cases an amaurotic loss of the pupil reaction is seen. The extrinsic eye muscles are always intact. In this respect this poisoning differs from the similar picture in meat poisoning. Less well known is perhaps that clinical picture described by Louis Ziegler in 1920. In Philadelphia society a number of young society women were attacked by an insidious atrophy of the optic nerve. Over a long period they had taken eau-de-Cologne poured on pieces of sugar. The American scents at that time contained 25 per cent. of methylated alcohol. These infinitesimal doses were not sufficient to produce the common poisoning picture. Where the Wassermann reaction is negative and tumour may be excluded in a chronic optic atrophy, it may therefore be of interest to examine for formic acid in the blood and urine. It is also necessary in cases of chronic retrobulbar neuritis to think of poisoning with lead or carbon disulphide, especially in cases of concentric shrinkage of the field. I pass over the acute quinine amaurosis in women endeavouring to cause abortion.

DEFICIENCY DISEASES

Hemeralopia must of necessity be connected with a retinal nutritive disturbance—not yet quite detected. Here I exclude anatomical retinal diseases such as retinitis pigmentosa or siderosis. Only during the last few years has the cause of this rather obscure condition been found. It is due to a lack of vitamin A. This circumstance explains why cod-liver oil has such a good effect, and why the spring fast-time predisposes to the outbreak of the sickness. Such night-blindness is followed, more or less, by xerosis conjunctivae. Suitable diet cures the trouble.

Much more dangerous is keratomalacia in infants. It was the great paediatric star of Breslau, Czerny, together with Keller, who introduced the term "*Milchnährschaden*." By this is meant that the infant is supernourished on carbohydrates. It is all right for three to six months, or even longer. Then a conjunctivitis suddenly appears. To begin with it does not look dangerous. But an observant surgeon will even then notice the white spots near the limbus, described by Bitôt. This xerosis rapidly spreads, and if the case is not properly treated necrosis of one or both corneae may occur. In Norway this disease is seldom observed. In Denmark it was relatively frequent during the war. Here it was described by Professor Block and Edmund Jensen. But its real cause was first found by Saunte, and later studied by Rönne and Blegvad. Supported by the findings of Monrad they have demonstrated the almost magic effect of sweet milk together with carrots and a little cod-liver oil. It may, though seldom, attack adults living on a diet poor in vitamin A. The aetiology of the phlyctenule is greatly disputed.

Some, such as Maitland Ramsay, hold entirely too bigoted a view that it is caused by a chronic digestive disturbance. Others hold, quite as certainly, that the singular cause is scrophulo-tuberculosis. One thing is sure. Bad hygiene plays a great part in its pathogenesis by preparing the soil. Wessely has emphasized that patients suffering from phlyctenular kerato-conjunctivitis are liable to catch catarrhs because of being supersensitive. However, the diagnosis of phlyctenular conjunctivitis must always remind the doctor that the child does not belong to a robust type.

OCULAR TUBERCULOSIS

A uveitis must always be regarded as a sign of a general disease when no external infection can be found. Because of that a uveitis—and also the much more infrequent scleritis—is always reason enough for a close internal examination. There is hardly any general disease which may not be the cause of it. But tuberculosis and syphilis are the predominant causes. The investigations of the last few years by Schieck and Werdenberg seem to have proved that ocular tuberculosis must not be regarded as a disease *sui generis*. It is to be considered as a metastasis in the eye. As a rule the glands at the hilus are the starting-point. As we do not know for certain what "rheumatism" really is, it is very probable that a number of cases of so-called "rheumatic" uveitis are of a tuberculous origin. The entire chapter of "rheumatism" has now to be recast. I may recall that a disease such as erythema nodosum was regarded as "rheumatismus acutus mitis cutis." Probably the general opinion is that it is a toxic skin affection in a tuberculous patient, of the same kind as von Pirquet's skin reaction. It is held that it appears simultaneously with the manifestation of a previously latent tuberculosis of the bronchial glands. But, on the other hand, we must remember that in a certain percentage the reaction of von Pirquet is negative. It is advisable to maintain the old cause in those cases. In the same manner we must still believe in "rheumatic" uveitis in a not insignificant percentage. In this group we also include the gonorrhoeal cases, and these must be considered as due to metastatic inflammation. Thus any violent iritis in an adult—especially combined with pain in the joints—ought always to direct the physician's attention towards a Neisser infection as the cause.

This is not the place to go into the differential diagnosis between tuberculous and syphilitic iritis. The Wassermann reaction has greatly facilitated the diagnosis as regards syphilitics when the clinical picture is typical. On the other hand, it may be more difficult in all cases to be quite sure about the origin of a tuberculous iritis. Axenfeld and Stock particularly deserve the greatest praise for their minute experimental investigations of uveal tuberculosis. By injecting attenuated tubercle bacilli into the ear veins of rabbits tuberculous nodules were developed. The retina remained histologically free, just as in miliary tuberculosis. They showed also how the not uncommonly recurring haemorrhages in the corpus vitreum in young—especially male—persons were caused by a periphlebitis. Often they succeeded in thus demonstrating directly a tuberculous process. In others they had to suppose as cause a toxic effect. The most recent explanation by Meller, in regard to sympathetic ophthalmia, is that it is due to attenuated tubercle bacilli. He has supported this clinically as well as experimentally.

MISCELLANEOUS CONDITIONS

During later times the importance of the internal secretory disturbances in aetiology and pathogenesis have been minutely studied. The description of these examinations would call for an entire evening. I must restrict myself to mention of the vast importance of eye symptoms in the diagnosis of tumours in the region of the hypophysis. In diseases here the ophthalmologist will be the

best helper, not only to the physician, but also to the surgeon. An examination of the field of vision is always strongly called for where amenorrhoea exists without any "physiological" reason, or in impotence of a too early appearance. It is hardly too much to say that but for his ophthalmological helper Harvey Cushing would not have been able to build up his splendid technique as a brain operator.

It is still disputed whether the syndrome osteopsathyrosis idiopatica—fragilitas ossium—is due to an inherited endocrine disturbance. This ailment is characterized by the triad: persistent blue sclera, fragility of the bones, and early labyrinth deafness. In any case the school physician finding a pupil with blue sclerae must later on have his attention directed towards the ears and bones.

The importance of trauma as a releasing factor with luetic parenchymatous keratitis is still *sub judice*. At least so much is certain, that it is not merely the vagrant spirochaetes in the lymph streams in the corneal tissue which are the cause. If so, it would be supposed that an energetic antisiphilitic treatment would prevent an attack of the keratitis in the second eye. On the contrary, this is rather the exception than the rule. There must therefore be an unknown *x*. Supported by a very minute investigation, clinically as well as serologically—after the method of Abderhalden—Zederkreutz, Johanson, and Enroth have attempted to prove that an endocrine disturbance may be found in more than 60 per cent. of all cases of luetic parenchymatous keratitis. Investigations—particularly German ones—have demonstrated that about 90 per cent. of all cases of deep keratitis are due to lues, especially congenital. According to von Michel tuberculosis is the cause of the remaining 10 per cent. In our time of accident insurance it is certainly correct to accept Schieck's and von Szilly's opinion as to questions about the importance of trauma in keratitis parenchymatosa. Parenchymatous keratitis is due to two factors which act together—infection + disturbed metabolism. An incidental cause—such as a trauma—releases the disease. Before finishing my remarks on the disturbances of metabolism and consequent eye symptoms, I merely mention the great diagnostic value they have in Graves's disease.

M. Piani (*Thèse de Paris*, 1934, No. 29) states that in spite of the excellent climate, temperate winters, and mild autumns Corsica suffers considerably from three serious epidemic diseases—namely, malaria, undulant fever, and tuberculosis. The almost complete absence of hygiene, due to the poverty and lack of education of the native population, is to a large extent responsible for their spread. Malaria has always been prevalent in Corsica, especially on the east coast, where it has assumed alarming proportions. The average parasitic index for the whole of Corsica is 18.56, and the splenic index 38.84. These figures, however, do not represent the true state of affairs, as they are merely the result of investigations carried out in certain parts of the island. Undulant fever is also remarkably prevalent. In 1929 the annual average number of cases was estimated at 200, while in 1932 600 cases were notified. Corsica is one of the French departments which pay the heaviest tribute to tuberculosis. During the period 1925 to 1930 the deaths from the disease have more than doubled (140 deaths in 1925 and 291 deaths in 1930). The principal causes of the spread of tuberculosis are the almost complete absence of anti-tuberculosis services, the yearly return of a large number of Corsicans who have contracted tuberculosis in France, the ignorance of the most elementary rules of hygiene, and the failure to diagnose numerous cases which are mistaken for undulant fever or malaria. Seasonal diseases such as pneumonia and pleurisy are not uncommon, owing to lack of precautions against changes in the winter. As regards mental diseases, constitutional states such as idiocy, imbecility, and cretinism are the only conditions which are at all frequent.